Cardiac Arrest Through Volition

C. M. McCLURE, M.D., Lindsay

SINCE DISCOVERY of the heartbeat, yogis and fakirs have claimed to be able to control it at will, but there are no documented cases in the medical literature of cardiac arrest through volition, without physical manipulations of any kind. The following case is presented because it is unusual, perhaps unique.

REPORT OF A CASE

A 44-year-old aircraft mechanic of Danish descent was admitted to Lindsay Municipal Hospital, April 24, 1958, because of a cold with cough of two weeks' duration. He said that in the previous 20 years he had had six episodes of upper respiratory tract infection, and that during these periods he had found that by sitting quietly, relaxing completely and "allowing everything to stop," he could induce progressive slowing of the pulse until cessation of heart action would occur, then a feeling of impending loss of consciousness. After a few seconds of this sensation, he would take a deep breath and normal heart action would resume. These occurrences resulted in the patient's developing a fear of sleeping, lest his heart stop and not start again. In 1953 and several times afterward the author verified this story by auscultating the heart and palpating the radial pulse while the patient induced several seconds of cardiac arrest. At these times his color would become the ashen grey of sudden circulatory failure, and partial loss of consciousness would ensue. However, no cardiac irregularities were ever observed during either normal sleep or general anesthesia. Cardiac arrest occurred only when the patient deliberately induced it.

The patient stated that at the age of seven years he had had rheumatic fever, then was bedfast for a long time and took digitalis for five years thereafter. Since that time he had had a cardiac murmur but no arthralgia, dyspnea, orthopnea, fever or chills. He underwent tonsillectomy at age 12 and cholecystectomy in January 1958, both under general anesthesia, without incident.

Submitted October 30, 1958.

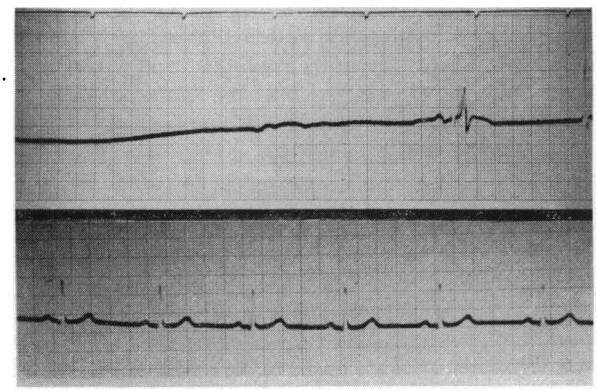

Figure 1.—*Above:* Electrocardiogram (lead I) showing cardiac arrest through volition. *Lower:* Normal tracing (lead I) for same patient.

On physical examination, the oral temperature was 99°, the pulse rate 60, respirations 18 per minute and blood pressure 134/74 mm. of mercury. The patient was muscular, intelligent, somewhat tense and anxious. There was no icterus, cyanosis or edema, but there was clubbing of all fingers. The only other abnormality noted in a complete examination was a grade I systolic murmur heard over the aortic area, becoming grade II along the left sternal border, the sound transmitting poorly to the apex. The rhythm was regular. Upon x-ray examination it was observed that the size and contour of the heart were within normal limits.

Electrocardiographic leads were connected and the patient was asked to induce slowing of the heart. This actually occurred with no physical manipulations except lying very quietly and allowing respiration to become quite shallow. The electrocardiogram showed slowing of the sinus rate progressively to the point of sinus arrest for a period of five seconds, followed by several atrioventricular nodal beats, and then resumption of sinus bradycardia at a rate of about 55. At several points in the record, occasional atrioventricular nodal beats were observed. An electrocardiogram an hour later with the patient at rest was normal.

A consultant who examined the patient and read the electrocardiograms suggested the use of sympathomimetic drugs, but subsequent use of atropine, ephedrine, amphetamine and aminophylline at different times did not change the arrest mecha-

nism. Even after recovery from the respiratory tract infection, the patient found that he could still induce bradycardia and brief periods of cardiac arrest almost at will.

DISCUSSION

It was felt that the clubbing of the fingers noted in this patient was familial, since it was present also in his son, who was healthy. The underlying cardiac change is believed to be well compensated rheumatic heart disease with aortic valvulitis. The bradycardia and cardiac arrest are probably manifestations of exaggerated vagotonia, induced through some mechanism which, although under voluntary control, is not known to the patient himself. Careful observation did not reveal any breath-holding or Valsalva maneuver in connection with the cessation of heartbeat. Apparently the patient simply abolished all sympathetic tone by complete mental and physical relaxation.

SUMMARY

A case is presented of a patient with old rheumatic heart disease, who is able to produce cessation of heartbeat, apparently by volition alone.

287 East Hermosa Street, Lindsay.

Acute Renal Failure Manifesting as Water- And Salt-Losing Insufficiency

AARON J. FINK, M.D., Mountain View

CLINICAL SYNDROMES involving primary inability by the renal tubules to retain sodium have reached their fullest expression under the generic term "salt-losing nephritis." The condition usually is chronic, requiring long-term salt replacement therapy. Except for the reversible urinary salt loss of the diuretic phase of lower nephron nephrosis and that caused by diuretic agents, relatively little has been written of transient primary renal salt-losing syndromes. Following is a report of a case of reversible acute salt-losing renal insufficiency occurring after nephrectomy.

REPORT OF A CASE

A 75-year-old white woman was admitted to the Highland-Alameda County Hospital on January 22, 1958, following a fall at home, after which she apparently lay on the floor all night, without voiding or eating, until found by a friend the next morning. She said that she had had no previous weakness, dizziness or imbalance and she was proud of being active. The last time she had needed the services of a physician was some forty years before for the birth of a child.

Upon examination it was noted that she was alert and spry-appearing. There was a small laceration on her forehead. The blood pressure was 104/60 mm. of mercury and the pulse rate 76 with a regular rhythm. The bladder was palpably enlarged above the pubis, and on catheterization 200 cc. of grossly bloody urine was removed.

There was motor weakness of the upper and lower extremities and assistance in walking was required. No neurological abnormalities were noted. X-ray films of the skull and chest taken at the time of admission were normal. The patient was then referred to the urological department where cystoscopy revealed the presence of what appeared to be diffuse, severe, acute hemorrhagic cystitis.

Submitted September 5, 1958.

In light of the patient's debility, an indwelling catheter was placed and laboratory examinations were obtained the next day. Results of centrifuged urine examination showed alkaline reaction, 2 plus albumin, no sugar, 140 leukocytes and 350 plus erythrocytes per high power field. Hemoglobin was 13 gm. per 100 cc. of blood, and leukocytes numbered 15,800 per cu. mm. Blood urea nitrogen was 100 mg. per 100 cc. and blood creatinine was 1.7 mg. per 100 cc. A urine culture grew an antibiotic resistant Aerobacter aerogenes. Phenolsulfonphthalein excretion was 5 per cent in the first hour and 35 per cent in the second hour.

The clinical impression was that the patient had acute and chronic hemorrhagic cystitis superimposed on probable carcinoma of the bladder with bilateral hydroureter. However, less than a month later, following rehydration, the blood urea nitrogen was down to 11 mg. per 100 cc. The hemoglobin content eventually became stable at 8.8 gm. per 100 cc.

An electrocardiogram and an x-ray film of the chest were interpreted as being within normal limits. Intravenous pyelograms obtained at this time revealed the presence of mild bilateral pyelectasis and caliectasis with a strong suspicion of a filling defect in the right renal pelvis. A cystoretrograde study carried out February 25, 1958, showed considerable resolution of the previously noted inflammatory process in the bladder, and there was no evidence of carcinoma. In differential phenolsulfonphthalein studies of the kidneys, there was excretion of 1 per cent of the dye from the right side and of 10 per cent from the left in 12 minutes. Bilateral pyelograms showed a ragged, irregular filling defect involving the right renal pelvis and associated caliectasia. Inflammatory changes involving the left upper ureter were also noted. Microscopic examination on culture of specimens of urine collected in a 24-hour period were negative for acid-fast organisms. A retrograde study a few days later again showed the irregularity of the right renal pelvis, consistent with the impression of right renal pelvis